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Art. I.—*On the Pathology of Yellow Fever.* By Josiah C. Nott, M. D., of Mobile, Ala.

Rational practice can only be based on sound Pathology, and my object here is to give *facts* derived from bed-side and *post mortem* observations.

A very fatal epidemic of yellow fever prevailed in Gibraltar in 1828, from the middle of August until the 25th of December, a period of about four months and a half. The French government, desirous of collecting precise information respecting this disease, appointed a commission composed of MM. Louis, Trousseau, and Chervin, all gentlemen of the highest professional standing, for this important object. They proceeded to Gibraltar, where they arrived on the 23d of November, *only* 33 *days before the termination of the epidemic.*

The commission performed its duty ably and faithfully, and with the assistance of the eminent English and Spanish physicians, then in Gibraltar, viz.: Mr. Gillkrest, Fraser, Mery, Dias and others, collected a mass of most valuable and interesting facts.

It is a well-known law of epidemics, that they differ not only in their symptoms, but in their pathological characters in different years and places, and M. Louis, who has published a masterly analysis of the facts collected at Gibraltar, says: "I would beg the reader to remember that this work is not a *treatise* on yellow fever, but a history of the epidemic yellow fever which prevailed in Gibraltar, in 1828. *All* the general facts which result from my analysis *may not be found in other epidemics.*"

Now, although this work is put forth as the history of a small portion of one epidemic, many (overlooking the various grades and shapes which the same disease will assume at different epochs) have strangely received the facts and deductions of M. Louis as fixed and invariable laws.

M. Louis has analyzed his limited and imperfect facts with all that ability

and honesty of purpose which so eminently characterize him; but any one who has had much experience with this disease and studied its phenomena, will see at a glance that his facts were too limited, his premises often false, and his deductions consequently erroneous—but still the work is full of instruction and of incalculable value as a model for similar investigations.

I have witnessed five epidemics of yellow fever in Mobile, viz., in 1837—'39,—'42—'43 and '44, each of which has presented some predominant peculiarity of type, and all demanded some modification of treatment. My medical friends and myself have made autopsies each year, but I have preserved detailed notes of the last two only—the dissections of the previous years, however, lead to the same general conclusions.

Excluding all doubtful cases, I have 16 autopsies remaining, and it so happens that they are equally divided between the two years 1843 and 1844. The dissections were made by Drs. Ross, Crawford, Gale, Lewis and myself. I assisted in all except two or three. The patients presented the usual train of symptoms during their illness—the skin was yellow in every case, and both stomach and bowels contained the characteristic black matter in all.

It will be remembered that M. Louis' conclusions are based on the dissections of 23 cases, all of the same epidemic. A peculiar change in the liver was the only constant lesion in his cases, and this he considers as *essential* to the disease, and a *diagnostic* sign. But there being no change in the liver beyond that of *colour*, which he regards as insufficient to account for the death of the patients, and all other lesions, gastritis, &c., being often wanting, he says we must look to the *cause* of the disease and not to appreciable pathological changes to explain the result. In other words, death results from the introduction into the system of a subtle *poison*, whose mode of action is unknown to us.

The facts which I am about to present, prove conclusively that this peculiar lesion of the liver is not more constant *in the yellow fever of Mobile*, than other lesions, and it is worthy of notice that there is a marked difference in the frequency of this lesion in different epidemics here.

M. Louis describes the liver as being in an anæmic condition, pale, dry and friable—the colour of straw, fresh butter, sole leather, *café au lait* or mustard, and in no instance as dark or darker than natural. I will now proceed to give my own observations.

Liver.—Of 8 cases dissected during the epidemic of 1843, in Mobile, the livers in 2 only corresponded with the description of M. Louis—they were pale, and when torn resembled very closely ginger bread or new leather; and the 6 others were of a dark blue or dark chocolate, presenting different shades of colour, and instead of being dry, they were excessively engorged with blood; the latter cases correspond with the description given by Dr. Hulse, of the cases dissected in the Marine Hospital at Pensacola in 1841.

Of the 8 dissections in 1844, the livers in 4 corresponded with the description of Louis; 2 were of a dark olive, and 2 were perfectly natural.

Taking the whole 16 livers collectively, 6 were some shade of yellow, dry and friable; 2 olive; 2 normal, and 6 darker than natural and much engorged. There can be no error in these facts, for Louis' liver was our standard for comparison in every case.

M. Louis has, I believe, never lived in a bilious or yellow fever region, and has fallen into another error in supposing this liver to be peculiar to yellow fever—I have repeatedly seen facts to the contrary, and two of them have occurred this summer (1844)—one a man by the name of Sandy White, who was walking about during the day, and died at night with violent pains in the bowels; poison was suspected, and the body was opened by several physicians. The other was a Frenchman, who was brought from three miles in the country into the city hospital, dying with protracted bilious fever.

In both these cases the straw-coloured liver was found, without any trace of black matter in the stomach or bowels, or other mark of yellow fever; the Frenchman had a yellow skin, but this is common in other fevers.

Setting aside then the liver, the leading facts and conclusions of M. Louis are strongly corroborated by my observations.*

Gall Bladder.—This was found in all the sixteen cases, except one, to contain bile, varying in quantity from ℥ss to ℥iv; colour from pale green to olive, and even black; consistence from water to tar—in one case the cyst contained about ℥iv of colourless fluid, resembling gum water or mucus.

The secretion of bile in this disease is almost invariably suppressed early; in severe cases it is rarely vomited after the second day, and I believe I have never seen it after the third day when they were fatal, except in one or two very protracted cases.

Stomach.—Of the eight cases in 1843, this organ in four presented no appreciable morbid change; the mucus coats throughout were free from injection or extravasation of blood, smooth and of normal thickness; consistence good, giving strips from four to twelve lines. In the other four cases the mucous coats were more or less red, thickened, mammilated and softened.

* Since writing the above, I have received from France a Report made to the Academy by M. Chervin, on a memoir of M. Rufz on yellow fever, and I am gratified to see that my observations on the liver are here confirmed. M. Chervin states that he has made five hundred autopsies after death from yellow fever, in the West Indies, Europe and America, and draws the following conclusions:—

"1st. That the lesion pointed out by M. Louis as constant in yellow fever, is often wanting, and that it is not rare to meet the liver in its normal condition in this disease.

"2d. That this organ shows not unfrequently a yellow colour in the remittent and intermittent fevers of hot climates, and that consequently the anatomical lesion, cited by M. Louis, is not *characteristic* of yellow fever, and that therefore the deductions which this physician has drawn from it fall of themselves."

M. Chervin also shows that M. Rufz' dissections do not bear out M. Louis, although they have been quoted with this view.

Of the eight cases in 1844, the mucous coat of the stomach in three were perfectly healthy, and five presented, as the above, various morbid appearances; but in no case was the mucous coat ulcerated or reduced to a pulpy consistence. All the stomachs contained black vomit.

Louis found the stomach normal in five out of twenty-three cases; in a portion of the others the morbid changes slight, and taking the whole together, the lesions in the stomach were not greater than are found in deaths from other acute diseases in Paris. I would make the same remark of yellow fever in Mobile.

Intestines.—My professional engagements would not permit me to examine every inch of the intestinal canal with the minuteness of MM. Louis and Trousseau, nor have I deemed it so important, as the great points of doubt and dispute are the *liver* and *stomach*, which were faithfully examined; the facts, however, as far as they go, may be relied on.

In every case the small intestine contained matter resembling the black vomit very closely, but usually more pasty and tenacious. With the exception of two or three cases, the large intestine contained it also; here it was frequently dryer, resembling sometimes pickled walnuts pounded, and sometimes in black scybala—in some cases the black pasty matter was found plastered over the whole surface of the small and large intestines, but the black matter is more constant the nearer you approach the stomach.

In one case the whole canal, large and small, was coated with a thick, tenacious matter, of a dark purple colour, like blackberry jam—a compound evidently intermediate between blood and black vomit—this I shall show hereafter was blood in the *transition state*, gradually turning black from the action of acid in the secretions of the intestines.

M. Louis saw black matter in the large intestine when it did not exist in the small intestine or stomach, but I have never met with a similar instance.

I have repeatedly seen patients (which I had no opportunity of examining after death) vomit or purge large quantities of fluid blood, and in some of the cases the black vomit was distinctly mingled with it, as in the case of Mr. Covert, whom I saw with Dr. Mordecai.

Though seen in some cases, traces of inflammation were less frequent and decided in the intestines than in the stomach; the small intestine rarely showed softening or thickening of its mucous coat; softening in the large intestine was very common, but being most frequently unaccompanied by thickening or redness, it could not be regarded as the result of inflammation. The morbid appearances met in the intestines are so common in other acute diseases, that much importance cannot be attached to them.

Bladder.—The bladder, in every case, contained urine, from a few ounces to two pints. In three cases the urine was bloody, in two it contained black matter like the black vomit—in the others the urine was healthy or varied slightly from it—mucous coat generally normal, but in some cases highly injected.

The heart in some cases was found softened and flabby. The brain, kidneys, spleen, lungs and other organs, presented no lesions sufficiently constant to give them importance. Rigidity of the muscles always took place after death.

Blood.—This was found dark and fluid in every case when the bodies were opened; minute observations were not made in all the cases, but in about one half, the blood was collected by thrusting a trocar into the right auricle and drawing it off into clean quinine bottles; it was so fluid, and was accumulated in such quantity in the auricle and veins connected with it, that 10 or 12 hours after death it would run freely through the canula to the amount of a pint or more. When set aside it coagulated at intervals, varying from 15 or 20 minutes to 36 hours; the clots were soft, grumous, easily broken down, showing a great deficiency of fibrin, and corresponding with Andral's description of the blood in the other pyrexiæ. In one case it did not coagulate at all, but presented a true state of dissolution.

In one case only the blood coagulated promptly and firmly like inflammatory blood; this was in a patient who lingered eleven days, and who for three days previous to the last two, I thought would recover. We found in him well marked inflammation of the peritoneal coat of the large intestine, and also of the interior of the pericardium where were deposited coagulable lymph and bloody serum. These appearances of fibrinous blood, according to Andral and Gavarret, may be accounted for by the supervention of this acute inflammation, as I shall point out farther on.

Black Vomit.—There have been many speculations on the nature and formation of this fluid, all of which are unsatisfactory; they are well known to the profession, and I shall here merely state my own opinions and the facts on which they are based.

It cannot, I think, be a secretion, because it is most commonly seen in little particles or masses of various magnitude which could not pass through a secreting capillary, and my own opinion is that the black vomit is *blood*, exhaled in its natural state from the capillaries of the stomach, intestines and even the bladder, and changed black by the secretions with which it comes in contact; this chemical change, my facts go to show, is produced by one or more acids.

With the assistance of my friend, Dr. P. H. Lewis, I have tested the black vomit in a considerable number of cases this summer, (1844) and in every instance I have found it to be acid; when ejected from the stomach during life, it invariably turned litmus paper red, and the aqueous portion of that which was taken from the stomach after death and filtered, in several cases effervesced strongly with carbonates. The aqueous portion thus filtered, differed in colour; in some it was perfectly limpid like water; in one of a light green colour like dilute bile with an acid added, and in others, it was of a deep brandy or rum colour; which appearance was no doubt given by a small admixture of blood.

The secretions of the stomach in yellow fever, are often excessively irritating, and this property is probably attributable to the presence of acid; the patient often complains in the black vomit stage of a burning or scalding sensation in the stomach which is immediately relieved by throwing off its contents. The patient, too, often complains of the black vomit scalding the œsophagus, which, after death, is usually found more or less denuded of its epithelium. The acridity of this secretion may possibly account for many of the morbid changes in the stomach and œsophagus. A morbid secretion of tears will scald the cheek; mucus from the nose, inflame the lip; morbid secretions from the bowels excoriate the *anus;* morbid bile irritates the stomach and bowels, &c.,—and we know that the gastric juice will often corrode the stomach in a short time after the extinction of life.

The next step was to ascertain whether acids would with blood produce a compound with the characters of black vomit. I accordingly took a few drachms of blood from the heart of a patient dead of yellow fever, and added to it 4 or 5 drops of muriatic acid, diluted with a drachm or two of water, and shook them well together; the black colour was produced instantly. The same experiment was tried repeatedly on the blood of yellow fever patients, and on that drawn from a patient with pleurisy by cups, and the effect was invariably the same.

Any one wishing to form a correct idea of black vomit, has only to treat blood in this way, and add a little gum water or flax seed tea to represent the mucus of the stomach, and his curiosity will be gratified; no one can tell the artificial from the genuine black vomit.

Sometimes the blood, after passing through the exhalents minutely divided, is coagulated in little particles, which, when blackened, present the appearance of coffee grounds; this appearance is difficult to imitate in the artificial black vomit, because we cannot readily produce these small coagula. I presume other acids will produce the same effect as the muriatic and crystallized acetic [citric?], the only acids which I experimented with.

We have then established two important links in the chain; the black vomit in the yellow fever of 1844 was acid, and acids turn the blood black.

Whenever, in yellow fever, blood is exhaled from the mucous coat of the stomach or bowels in small quantity, a *quantity proportionate to the secretions of these surfaces, it is (according to my observations) invariably found black, and the aqueous portion limpid or clear green.* If there be a slight excess of blood (more than enough to neutralize the acid) instead of black, we find a nut brown, a chocolate or reddish matter, and the watery portion, when filtered, of a rum, brandy or red colour. If the hemorrhage be great, we have (as I have often seen vomited) a fluid with all the characters of blood, either with or without a mixture of black vomit. I have often seen a tablespoonful or two of the "coffee grounds" at the bottom of the basin with a pint or more of pure blood; this I have several times pointed out to others. In the case of Mr. Covert, whom I saw with Dr. Mordecai, I

had presented to me at one time three basins, each containing a full pint of blood with the black vomit intermixed and lying at the bottom.

As an additional proof that the black vomit is blood, changed by the secretions of the stomach and bowels, I will state that I have never seen red blood, in yellow fever, tangled with mucus; when thus mixed it is always black.

This exhalation of blood and chemical change is by no means peculiar to the stomach, but evidently takes place over the whole mucous surface of the canal. I could cite many facts to prove this, and abundant evidence will be found in the work of M. Louis.

I have frequently seen black matter, like black vomit, in the urine, and it is formed no doubt in the same way, by blood combined with an acid in the urine, or in the mucus of the bladder. Dr. Lewis and myself are at this moment attending a medical gentleman (Dr. Fletcher) in whom this formation has been going on for some days, and at the same time the patient is throwing up black vomit and purging pure blood from the bowels.

There are many facts connected with other diseases, and analogies which would throw light upon this interesting subject, but I must touch them lightly for want of space.

Blood which is vomited or purged in other diseases, after being retained in the alimentary canal (in contact with the secretions) is usually very dark and not unfrequently black. In hæmoptysis the blood, on the contrary, is usually *florid*, and we are told by many that in the one case the blood is venous, and in the other arterial; but the blood in the two cases is doubtless exhaled from the same set of vessels, and the difference can only be accounted for by the chemical action of the gastro-intestinal secretions.

We know that in the vomiting of pregnancy, little specks or streaks of blood are frequently thrown up, and when females in this condition are attacked with fever of any kind, accompanied with excessive and protracted vomiting, small specks or streaks of black matter, like broken-up butterfly wings, are frequently seen. This kind of vomit is the usual precursor of genuine black vomit, and has the same explanation. Every experienced physician must have seen this, and my friend Dr. Crawford yesterday related to me a very interesting case of this kind attended by himself and Dr. Mordecai; the patient (the wife of a distinguished lawyer) during the month of December last, ill of a protracted fever, possessing no other symptom of yellow fever, and at a time and place where this disease was not prevailing, threw up black matter profusely, which could not be distinguished from the black vomit of yellow fever. We all know how abundant is the formation of acid in delicate females when pregnant.

A very small quantity of blood oozing gradually in a minutely divided form, and mingling slowly with the secretions of the mucous membrane of the stomach and bowels, will make a large quantity of black vomit. Judging from my experiments, I should think a tablespoonful would make a pint.

A moderate quantity of bile may exist in the black vomit without being perceived; this I proved by adding bile to the artificial black vomit, and by filtering the genuine black vomit, the aqueous part of which in one case was green, and this colour, I presume, was attributable to a small admixture of bile. Authors have gone into laboured descriptions of the *varieties* of black vomit, but my belief is, and the preceding facts go to prove that they are essentially the same, blood,—acids, mucus and aqueous fluid, mixed in various proportions. Give me blood, muriatic (or, I presume, any other acid) and gum water, and I will make it to suit the notions of the most fastidious pathologist: perfectly black, brown, reddish, &c.

We have thus passed in review the principal pathological changes seen in yellow fever, and we have found no one which is constant, no tangible cause of death. There has been a morbific cause at work; but we may ask with Louis, through the medium of what system does it exert its influence on the economy? Is it through the nervous system—is it through the blood?

Much ingenuity and labour have been spent in seeking a local habitation for this and other fevers, but all to no purpose. The well-informed and leading pathologists of the present day, Andral, Chomel, Louis, Alison, Tweedie, Watson, and, in fact, almost all except a remnant of the Broussais school, have abandoned this idea, and now regard the pyrexiæ and phlegmasiæ as distinct classes of disease.

The late work of M. Andral on the blood must be esteemed one of the most valuable pathological contributions of the day, and leads the way to important results. The old humoral pathology, though cumbered with much fanciful speculation and false assertion, contained such obvious truths as to have numbered amongst its advocates most of the great names which have graced the history of medicine from Hippocrates to the present day. *Facts* were noted which could not be explained, because organic chemistry had not reached a sufficient degree of accuracy for this kind of analysis. But chemistry is now marching up, and demonstrating to be true what before was mere probability. Besides Andral and Gavarret, many other of the French as well as German pathologists have, by actual experiments and analyses of the blood, already brought to light truths of the greatest interest and practical importance—some of these I will notice briefly.

M. Andral, in his recent work alluded to, uses the following bold and striking language.

"The pyrexiæ form a large class of acute diseases which it has been vainly sought to dismiss from nosological systems, in order to throw them into the order of simple inflammations. Such pretensions, however, cannot be maintained: the pyrexiæ exist as diseases apart. The causes which often develope them, the symptoms which characterize them, the *special nature* of the alterations that they produce in the solids, the epoch of development of these alterations (often posterior to that of the febrile movement)

are sufficient reasons for not confounding the pyrexiæ and the phlegmasiæ; but the *analysis of the blood* comes still more strongly to establish a very remarkable difference between the two classes. The results furnished by this analysis have something so marked, that they seem to me to fix in a definite manner, the distinction vainly combated, between the pyrexiæ and the phlegmasiæ."

Now what the morbific cause *is*, what its *modus operandi*, what the nature of all the morbid changes in the system, we cannot now, and probably never shall be able to discover; but although we may not be able to tell what the essential nature of yellow fever is, it is not the less important to prove what it *is not*—we may thus strike out a fatal error, as the treatment depends so much upon the pathology.

The following are the normal proportions of the blood in health, as laid down by Andral after a very extended analysis—1000 parts contain 3 of fibrin, 127 of globules, 80 of solid matter of the serum, and 790 of water. These ingredients bear a certain relative proportion to each other, which alone are compatible with health; each one may fluctuate between certain limits without indicating disease, but whenever these fixed bounds are passed, *disease is invariably present.*

The average of fibrin, as stated, is 3 in healthy human blood, and may range from 2.5 to 3.5 in health—there are extreme cases where it has been seen as low as 2 or as high as 4.

The average of globules in healthy blood is 127, the minimum 110, the maximum 140; but this maximum is linked with plethora, which becomes a true morbid state.

The solid materials of the serum, which are composed almost exclusively of albumen, have 80 for their mean, which has also its maximum and minimum. There are also smaller quantities of the salts of soda, potash, lime, magnesia, iron, colouring matter of the bile, &c.

The proportions of all these ingredients play very important parts in other diseases, but my present object is to establish a distinction between the phlegmasiæ and pyrexiæ, and I shall confine myself to this point.

After eighteen hundred careful and well-directed experiments, M. Andral lays down the following important and invariable law: viz., *that in local inflammations (or phlegmasiæ proper) there is always an increase of fibrin in the blood, and that the true buffy coat is never seen unaccompanied by local inflammation.**

From 3.5, the maximum of fibrin in health, it is increased during inflammations to 6, 7, 8, and sometimes as high as 10, while in fevers it is diminished to 2, to 1, and even to .9. In the phlegmasiæ this excess of fibrin continues as long as the inflammation persists, and in spite of repeated and copious depletion.

* There are *apparent* exceptions in anemia and pregnancy, which will be found satisfactorily explained in Andral's book.

At the *onset* of idiopathic fevers, and in those of light grade, the fibrin is not invariably diminished; but if the disease continue with any intensity, this diminution always occurs.

When the pyrexiæ are complicated with local inflammations, as pneumonia, hepatitis, &c., there is an increased production of fibrin, but it can never be increased so much as in simple inflammations, because the morbific cause of the fever is counterbalancing it by a contrary tendency. It is a fact, too, that those local complications which *belong* to the pyrexiæ, and which are *secondary effects* of the morbific cause, for example, the eruption of small-pox, the intestinal ulcers of typhus, buboes of plague, the gastritis occasionally seen in yellow fever, &c., are not sufficient to cause an increase of the fibrin of the blood.

All the tissues obey the same law; the increase of fibrin is invariably seen in inflammations of the nervous system, the muscular-parenchymatous structures, in the serous and mucous membranes, &c.

"The diminution of the fibrin, every time that it takes place, produces remarkable modifications in the physical qualities of the blood. Whatever be the pyrexiæ in which it exists, the blood drawn from a vein presents the following condition. The serum and the clot are imperfectly separated from each other, whence it follows that there *seems* to be but little serum in proportion to the clot. The clot is voluminous; it often fills the whole breadth of the vessel in which the blood has been received; it is never elevated upon its borders, as is so commonly the case with the clot of the phlegmasiæ. Its consistence is always slight, it is torn and broken with the greatest facility, and there are some cases even where, by the slightest pressure, it may be reduced to a true condition of diffluence; it ceases then to form a single mass, and is divided into a number of grumous portions which mix with the serum, and colour it of a more or less deep red. This is the condition of dissolution of the blood, so well described by the ancients, and which ought to be regarded as a necessary consequence of the diminution that the fibrin has undergone. Then, in effect, the net-work which maintained the globules pressed one against the other, and which, by its power of contraction, squeezed out the serum, no longer exists but imperfectly; thence also arises the great size of the clot which is found to be in an inverse proportion to its density, and which is not a certain index of the quantity of solid material that it contains." The size of the clot is also often increased in the early stage of the disease, by the large proportion of red globules.

These appearances of the blood exist in a remarkable degree in yellow fever, and are sufficient, without minute analysis, to point out a peculiar state of the blood, distinguishing it from inflammatory diseases.

In the phlegmasiæ the clot is smaller, more dense, and of superior consistence, and if the blood has been *properly drawn*, the clot will be covered by a buffy coat of variable thickness, and is usually cupped. Except in anæmia, and, perhaps, occasionally in the last stage of pregnancy, the buffy

coat invariably denotes a state of inflammation—no exceptions were found in eighteen hundred cases. The buffy coat may be *prevented* by improper manner of drawing the blood, but it cannot be *produced* where inflammation does not exist.*

The hemorrhages which occur in yellow fever, are not less conclusive as to the non-inflammatory character of this disease. The blood in malignant fevers, scurvy, purpura, and all those diseases in which passive hemorrhages occur, show, on analysis, a deficiency of fibrin in the blood—it is so constant that the absence of fibrin and hemorrhages *must be regarded as cause and effect.*

M. Andral, says: "Amongst the phenomena which seem to me to have a direct connection with the diminution of the fibrin, I believe I ought to place in the first rank the hemorrhages that are so common in all the pyrexiæ in which the adynamic or putrid type predominates; that is to say, those in which it has been *proved by analysis*, that the blood had lost some of its fibrine. It would seem that, in this condition, a certain diminution of the fibrin of the blood has, for effect, to permit the globules to abandon more readily the vessels which contain them."

"How, again, can we fail to remark the coincidence, so frequent in the pyrexiæ, of the diminution of the fibrin of the blood and of the facility of the production of those congestions, or sanguine states, *which have been so often confounded with true inflammations?* Wherefore this coincidence? Deprived of the ordinary quantity of fibrin, borne along with them in the torrent of the circulation, are the globules at the same time deprived of a means of regularization for their movements, and do they come in this way to *accumulate* and *stagnate in the capillary rete?* I know not, but it must be that there is some connection of cause and effect between the diminution of fibrin of the blood and the production of these congestions, since these latter follow so constantly the former."

Independent of the convincing proofs drawn from the blood, the symptoms of yellow fever, and the *post mortem* appearances which I have detailed, are sufficient of themselves to establish a marked difference between the two classes of disease. Acute inflammations of the abdominal and thoracic viscera (the brain is involved in more obscurity) are almost invariably declared by strong diagnostic symptoms during life, and, when fatal, leave behind traces too uniform and evident to be mistaken. Suppose you are called to

* When the blood flows *slowly*, the *buff* is not properly formed, because the slow discharge gives one portion time to coagulate before another, and only the blood last drawn furnishes the fibrin at the upper part of the vessel. Again, in a deep narrow vessel the buff will form much more decidedly than in a broad shallow one; because the thickness of the fibrinous crust will be greater. If the blood be agitated during its coagulation, the globules are mixed up with the fibrin, and the crust is imperfect and soft. In order, therefore, to test the blood fairly, it should be drawn in a full stream and received in a deep, narrow vessel.

a simple gastritis, enteritis, hepatitis, pleuritis or other acute inflammation, sufficiently violent to destroy life in from 3 to 6 days, (a common duration of yellow fever,) could you doubt about the diagnosis, or would you be disappointed in finding, after death, lesions sufficient to account for the termination? These are too plain to be disputed, and we are all agreed about them. But in yellow fever where shall we find the footsteps of the disease?

The *pulse* alone in yellow fever is sufficient to prove that the fever does not *originate* in gastritis; the pulse in the first stage of this disease is often full and bounding; and does not every pathologist know that gastritis is characterized by a contracted and usually very feeble pulse? In this stage, too, irritability of stomach, one of the surest signs of acute gastritis, is wanting.

The *stasis* of the blood in the small vessels, so well described and explained by Andral, no one in the present day will contend is evidence of inflammation; this fulness of the vessels of the mucous coat of the stomach is often seen in those who die of other diseases, or in those who have been hanged or destroyed by accident. Broussais has done much towards the advancement of pathology, though, like most medical reformers, he has pushed his peculiar notions to extremes. This branch of late years is much better understood, and old errors are fading away. Softening of the stomach has been shown to be an equally fallacious test of inflammation.

It has become the fashion of late to speak of yellow fever becoming "*localized*," but to my mind it is just as rational to say, that a storm has "*localized itself*" because it blows down a tree here and there, whilst it is shaking the whole forest to its foundations. That the yellow fever does, in the course of its progress, like all great epidemics, give rise to morbid changes in particular organs, no man of sense will deny; such effects are to be expected from all poisons which pass into the blood, or act in any way on the system. Sometimes in this disease there is gastritis; the liver is often changed in a remarkable degree; the kidneys are found altered in colour and texture; the lungs show dark patches; the brain is congested; bloody tumours form; the secretions are checked; the texture of the skin so changed that I have seen (as in the case of Capt. Atwood, last summer) the cuticle, in attempting to cleanse the face with a wet towel, wiped off, and the skin left as raw as a blistered surface, and this too 12 hours before death.

Who would say that small-pox and plague were *localized* because they effect the skin or glands; and so with the gastritis of yellow fever; nor is there any locality which can be assigned to inflammation which will account for the tendency to rapid collapse, the state of the blood, the depressing effects often seen from depletion, the early demand for stimulants, &c.

To my mind the preceding imperfect sketch of facts establishes satisfactorily the position, that yellow fever should not be ranked with the phlegmasiæ; but we must acknowledge that, though many important facts have been disclosed, there is still something to be explained, which the present

state of science cannot reach. We are here driven to analogies and probabilities, but it should be remembered that it is by rational theories we are most frequently led to the discovery of important truths.

Intermittent, remittent, continued and eruptive fevers are unquestionably produced by morbific poisons; we see effects which must have causes. Who can hesitate to regard yellow fever as a well marked-case of poisoning? Sometimes a patient, coming into the infected district, in perfect health, is stricken down by this poison with the rapidity of lightning; the powers of life are annihilated at once, and in 24 hours he dies without reacting from the first blow. I have seen several the last summer whose first symptoms were coma, or convulsions, or both. How different from local inflammations!

The poison is received through the atmosphere, but how or when the first impression is made, are points of dispute. Some poisons act on the nervous system; others make no impression until they are taken into the circulation. Now the poison of yellow fever probably acts in both of these modes; it may be carried by inhalation directly into the blood, changing its composition, and at the same time, while driven to every fibre of the system through the capillaries, may produce secondarily a deleterious influence on the ganglionic and spinal nerves. The muscular pains, the headache, chills, prostration, &c., at the onset prove that the nervous system is impressed, and the facts I have before given, show that the blood cannot be changed without a corresponding change in the various functions. We have seen that the changes in the blood are constant, but, on the other hand, we have no facts to prove that disturbance of innervation is the first link in the chain.

In reasoning on the laws of nature, we are stopped by ultimate facts beyond which we cannot look. Of the action of the nervous system in disease we know but little, and we are therefore forced to fall back on the *tangible alterations of the blood.* Even were we to admit the obvious changes in the blood to be the second link in the chain of causation, it is still no less a point worthy of our careful study. Vitiated blood, like gastritis, hepatitis, phrenitis, and other lesions which arise in the course of the fever, cannot be unimportant; it is to be studied and corrected.

The blood is the great fountain of life; it is itself a living fluid; the elementary particles of which have their attractions and repulsions, their vital affinities as well as their definite proportions, which are necessary to the maintenance of life and health: it presides over all the functions, and reason would say, and analysis proves, that whenever it is altered, health is disturbed.

Venous blood in the brain destroys life; animal heat is intimately connected with oxidation of the blood. All the vitality of the nervous and muscular systems seems to depend on the contact of the blood; this is proved by the effect of tying the blood-vessels, and also of diminishing the total

mass of the blood. When the blood is arrested in any part, this part loses its sensibility; the muscles are no longer subject to the will; they lose their characteristic property of irritability. When the brain is deprived of blood, death immediately follows. Venous and lymphatic absorption cease the moment the arteries are tied,—all nutrition, all secretion becomes abolished; gangrene occurs in parts deprived of blood; and when the circulation is arrested, *poisons no longer exercise their deleterious effects, &c. &c.*

The facts connected with the action of poisons, are interesting in relation to this subject, but I have space for but a few cursory remarks on this head.

Many of the inorganic poisons (as some of our medicines), are taken into the blood and emerge again from the body in the secretions, unaltered; they may produce changes beneficial or injurious according to the quantities taken and the condition of the system at the time; many saline preparations, acids, &c., are easily detected in the blood and secretions.

There are other inorganic substances which are not eliminated and thrown off from the system; but they form permanent chemical unions with the constituent tissues of certain organs, or with certain elements of the blood, and often produce changes incompatible with life.

The animal poisons—those which arise from contagious disorders, such as small-pox, scarlet fever, typhus, &c., are governed by laws totally different; they produce changes in the blood by which the same poison is not only reproduced, but multiplied in a remarkable degree. In small-pox, for example, the patient becomes surrounded by a poisonous atmosphere, and the virus is thrown out over the whole surface of the body in the form of pustules, each of which is capable of propagating the same disease to many individuals.

But there is still another class of mysterious poisons, included under the general term *malaria*, which holds the first rank in importance in our southern latitudes. These also are deadly in their effects, and governed by their peculiar laws, and long, patient and well-directed observations have brought a very large majority of our ablest pathologists to the conclusion that these poisons have not the power of reproduction in the human system, and consequently that diseases arising from these causes *are not contagious.*

Yellow fever stands first in importance in this class, but I shall not attempt to argue the question of contagion here. No one in or out of the profession, in Mobile, believes in its transmissibility, and our town is peculiarly well situated for investigating this point. Dr. P. H. Lewis, of our city, has been engaged for some time in preparing a history of this disease, and is now publishing, in the New Orleans Journal, a plain unvarnished statement of the facts, from which I think there can be no appeal.

Of the importance of the fibrin in the circulation of the blood, we are well assured by the important experiments of Magendie, from which it appeared that when the blood is in a great measure deprived of this constituent, or when its coagulating property is destroyed by the excessive or long-

continued use of alkalies, the *circulation of the capillaries can be no longer maintained, this preternaturally fluid blood being extravasated from them in various parts of the body, as in malignant fevers.*

The changes in the saline ingredients of the blood, afford an important field for exploration which has not been sufficiently cultivated. Stevens, and others have detected remarkable deficiencies in these ingredients. It is well known that the natural saline taste of the blood is often lost in fevers, and we have numerous instances recorded, where the blood has exhaled when drawn, an odour so fœtid, as to be offensive to the bystanders. Haller predicted a fatal issue in a case from this symptom alone. The effect of long-continued salt food in producing scurvy, is familiar to every one. Eating putrid flesh has produced typhoid fevers. On the other hand, acids, and particularly the mineral acids, turpentine, superacetate of lead, and all the salts, particularly those with excess of acid, have the effect of increasing the healthy *crasis* of the blood, and producing the opposite change.

The effects, too, of living upon much fresh animal food, in increasing the quantity of fibrin, in rendering the blood rich and abundant, and in disposing to inflammatory diseases, are familiar to all.

The researches of Gaspard and Magendie, in order to ascertain the effects of putrid vegetable and animal matter when introduced into the cellular tissue, or injected into the blood, further illustrate the importance that is to be attached to the morbid states of this fluid, as well as the nature and origin of various diseases. These physicians have fully proved that such substances, when thus employed, produce symptoms very similar to those of yellow or typhus fevers; and that after death, this fluid is found remarkably altered, being nearly altogether fluid, of a very dark colour, and partially exuded from the capillaries, both into the parenchyma of the viscera and from the mucous surfaces. That the blood is really altered in its nature by this inoculation, is proved not only by those changes, but also by the circumstance of its having lost its power of coagulating upon being removed from a vein soon after it has been thus injected, and by its speedy putrefaction.

The more recent experiments of Leuret and Hammont furnish like results; whilst others of Magendie show that dogs confined over, and breathing the effluvium proceeding from animal and vegetable matter undergoing decay, experience similar symptoms to those now referred to, and the same alterations of the blood, of the secretions, of the excretions and of the viscera, as observed in yellow fever: and in all these cases the morbid changes extend more or less to the soft solids, and *particularly to the mucous surfaces*, the lungs, the liver, heart, &c.; and in some of these cases, black vomit occurred.

M. Gendrin (so well known for his able works on inflammation and fevers), relates the following interesting and conclusive experiments. An ounce of blood drawn from the arm of a patient sick with putrid fever, was injected into the cellular tissue of the groin of a cat; he observed by turns

in the animal copious vomitings of bile, first yellow, and then green, dyspnœa; small, frequent and irregular pulse; brown dry tongue; progressive prostration, and, towards the last, convulsive movements. The following lesions were found after death; the skin of the groin was detached; the cellular tissue soft, pulpy, and of a yellow-ash colour; it had a fœtid odour and was covered with small red spots. The gastro-intestinal mucous membrane was in its natural condition; the blood was liquid and black; there was in the left pleura two ounces of dark serous blood; the heart was flaccid and soft; the dead body in a short time exhaled a putrid odour, &c.

Some blood furnished by epistaxis occurring in the same patient, was injected into the crural vein of a dog, who died with the same train of phenomena. He also injected the blood of patients affected with confluent small-pox, into the veins of different animals; violent symptoms supervened which were speedily fatal, and *post mortem* examination showed high inflammation of several organs.

MM. Depuy and Leuret introduced into the cellular tissue, and injected the veins of healthy horses, with blood from others affected with malignant pustule, and the disease was reproduced.

In conclusion, then, I would say that the foregoing facts establish—1st. That yellow fever should not be classed with the phlegmasiæ; 2d. That the blood is greatly changed from its normal state; 3d. That yellow fever is a condition of the system produced by a depressing poison which acts through the circulation; 4th. That the black vomit is blood, changed in colour by secretions, after it is exhaled.

It may now be asked what practical deductions are to be drawn from these pathological facts? And to my mind the answer is both easy and satisfactory. If yellow fever be the result of a depressing morbid poison, if it be not inflammatory in its nature, I would lay down as a *general rule*, that it is not a disease which demands *active* depletion, either by blood-letting or purging. According to the established rules of practice, the lancet is used to combat inflammation, plethora and active congestions, and where, I would ask, are these indications to be found?

We are, also, not unfrequently compelled to deplete actively in transient arterial excitement produced by stimulants, morbific miasms, &c., and where neither of the foregoing indications are present. Now this kind of excitement is occasionally seen in the yellow fever of Mobile, in the onset of the disease, when the system reacts from the first impression of the morbific cause; but such cases are rare.

If I am asked whether the lancet should be proscribed in yellow fever, I reply, certainly not. The poison of yellow fever (like that of the plague, scarlet fever, small-pox, &c., like opium, alcohol and other poisons), is sometimes followed by a turbulent and dangerous state of excitement in the arterial system, and the pulse *must be* brought down to its normal standard of force; but you must not expect to bleed a poison out of the system. When

the pulse is full and strong, and is decidedly *above par*, the lancet should be used promptly and boldly, to guard against the production of local lesions; but we should at the same time bear in mind that this stage is of short duration; that we are dealing with a specific disease which has a strong tendency to early collapse, and that we may soon have "need for all the strength we take away." These are cardinal points, which I take to be well settled in the minds of experienced and judicious practitioners.

In conclusion, I beg leave to remind the reader, that I am writing of the yellow fever of Mobile, as we have seen it during the epidemics and endemics of 1837—'39—'42—'43 and '44, which have afforded an ample field for observation. He who is ignorant of the various types in which this Protean disease appears in different years, and in different latitudes, must either not have read, or read to little purpose the history of yellow fever. In one epidemic, we are told the lancet is the sheet anchor—in another, it is death. This difference occurs to a limited extent in Mobile, but the *rule* is, beware of the lancet.

Even the blood-thirsty Rush, (whose philanthropy we all admire,) after singing so loudly the praises of the "*coup sur coup*" system in 1793, and pointing triumphantly to cases where he had drawn 100—150 and even 200 ounces of blood, is forced by stern facts to lower his tone in '98, and in 1805 he tells us, that many of his cases would bear but one bleeding, and others none at all, and this is the point to which experience and sober judgment must inevitably come.

Mobile, November 10th, 1844.

Art. II.—*Strictures on the use of the term Congestive as applied to low forms of Fever, with some general observations on the pathology of these diseases.* By Isaac Parrish, M. D. (Read before the Philadelphia Medical Society, at the session, 1844–45.)

The term congestive is now extensively employed to designate certain alarming, and oftentimes rapidly fatal symptoms which arise in the course of malarious fevers, more especially as they prevail in the southern and southwestern portions of the United States.

The fatality of these fevers, in certain seasons and localities, carries terror to the inhabitants, and the name by which they are known has become established in medical nomenclature as descriptive of a malady in which congestion of the internal organs is considered the most prominent pathological state.

I propose, in the present paper, to take exceptions to the term by which